Approcci interdisciplinari in reumatologia - 7ª edizione

MALATTIE REUMATICHE E DISORDINI ENDOCRINO-METABOLICI

TORINO
16-17 ottobre 2020

SESSIONE I – FLOGOSI E METABOLISMO (Moderatori: E. Fusaro, E. Ghigo)

14:30 La flogosi come fattore favorente di patologie cardiovascolari **(A. Benso)**

14:50 Fattori metabolici come determinante della risposta alle terapie **(G. Beccuti)**

15:10 Terapia biologica e con Jak inibitori nell'AR: effetti sul metabolismo **(M.C. Ditto)**

15:30 La gotta: diagnosi e terapia **(N. Ughi)**

15:50 Discussione plenaria

Fattori metabolici come determinante della risposta alle terapie

S.C. Endocrinologia, Diabetologia e Metabolismo U.
Dipartimento di Scienze Mediche
Università degli Studi di Torino

guglielmo.beccuti@unito.it

Cardiometabolic comorbidities in RA and PsA: lessons learned and future directions

Lyn D. Ferguson[1,2], Stefan Siebert[2], Iain B. McInnes[2] and Naveed Sattar[1]**

Ferguson - Nat Rev Rheumatol 2019

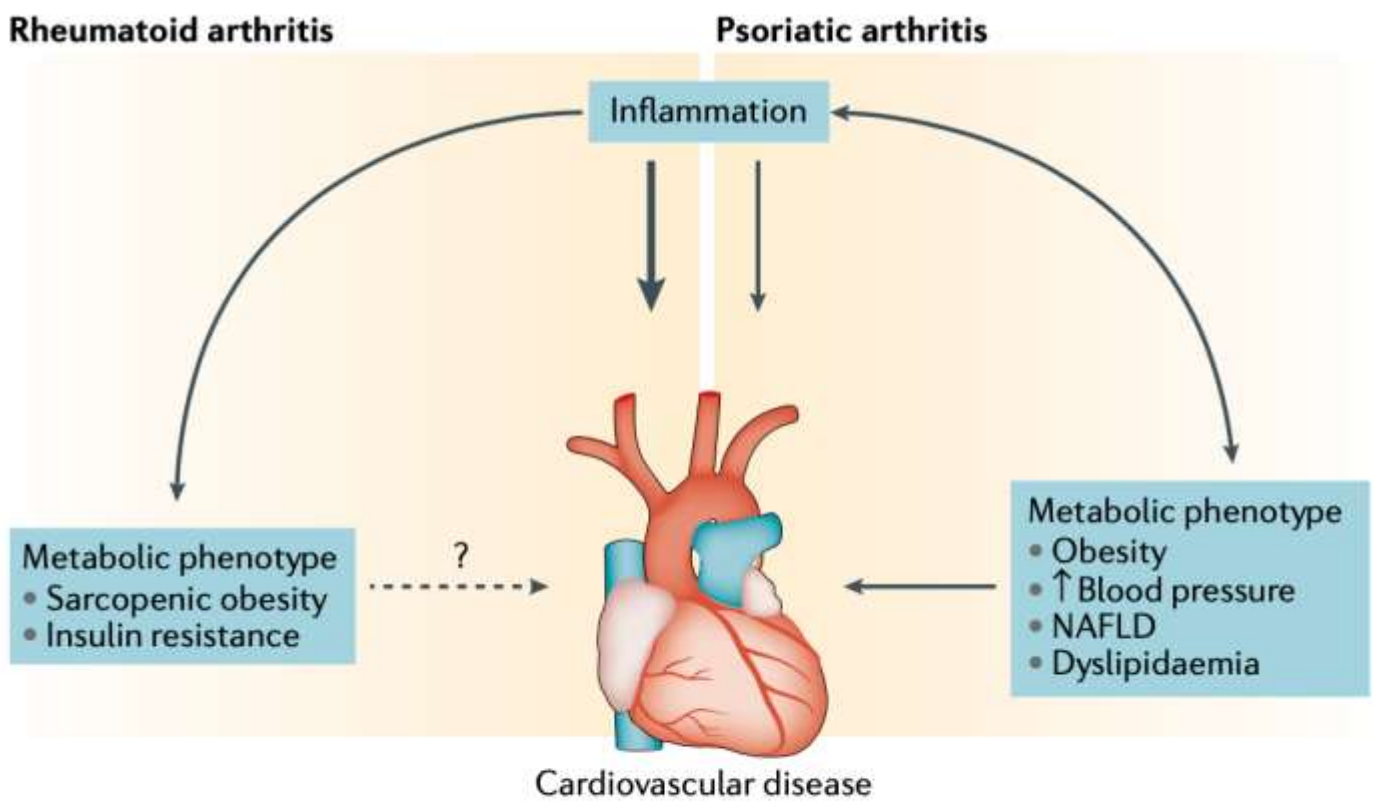


Fig. 2 | **Potential relationship between cardiovascular and metabolic comorbidities in RA and PsA.** Both rheumatoid arthritis (RA) and psoriatic arthritis (PsA) are characterized by systemic inflammation, which can contribute to an increased risk of cardiovascular disease (CVD) and metabolic disturbances. RA is associated with a high risk of CVD, probably owing more to chronic systemic inflammation than metabolic disturbances per se, but the impact of changes in body composition or insulin resistance on CVD risk is uncertain. By contrast, PsA is strongly associated with a dysfunctional metabolic profile (such as obesity and increased risk of type 2 diabetes mellitus), particularly in patients who develop the disease in later life, which might indirectly increase the risk of CVD in these patients. However, individuals who develop PsA at a younger age might have a larger inflammatory drive and a less obvious metabolic phenotype than patients with an older age of disease onset, and so inflammation might be relatively more important to CVD risk in these patients. NAFLD, non-alcoholic fatty liver disease.

Table 1 | **Cardiometabolic comorbidities in RA and PsA**

Cardiometabolic comorbidities	Rheumatoid arthritis	Psoriatic arthritis	Refs
Cardiometabolic outcomes[a]			
Risk of CVD	++	+	1,10,11,21
Obesity	+/–	++	4,73,74,79,84
Type 2 diabetes	+/–	++	92–95
Hypertension	+	+	23,102,103,105
NAFLD	+/–	++	98–100
Lipid profiles in patients with active disease[b]			
Total cholesterol	↓	↓	57,58
LDL-C	↓	↓	57,61
HDL-C	↓	↓	58,66

++ markedly increased; + increased; +/– mixed evidence; ↑ increased; ↓ decreased; CVD, cardiovascular disease; HDL-C, HDL cholesterol; LDL-C, LDL cholesterol; NAFLD, non-alcoholic fatty liver disease; PsA, psoriatic arthritis; RA, rheumatoid arthritis. [a]Compared with individuals of the general population. [b]Compared with individuals in remission or without disease.

Ferguson - Nat Rev Rheumatol 2019

The goal of therapy in treating RA e PsA

To improve health by preventing or treating metabolic complications

A primary focus on articular manifestation of RA e PsA

EXTENDED REPORT

Obesity is associated with a lower probability of achieving sustained minimal disease activity state among patients with psoriatic arthritis

Lihi Eder,[1] Arane Thavaneswaran,[1] Vinod Chandran,[1] Richard J Cook,[2] Dafna D Gladman[1]

Results Of the 557 patients included in the study, 36.2% were classified as overweight and 35.4% were obese. Overall, 66.1% of the patients achieved sustained MDA during the follow-up period. A dose–response association was found between obesity and the probability of achieving sustained MDA in the multivariate regression analysis. Patients in the higher BMI categories were less likely to achieve sustained MDA compared those in the lowest BMI category (overweight: OR 0.66 p=0.003; obese: OR 0.53 p<0.0001) after adjusting for potential confounding variables.

Conclusions Overweight and obese patients with PsA are less likely to achieve sustained MDA compared to those of normal weight.

↑ BMI: ↓ probabilità di stato di attività minima di malattia

Eder - Ann Rheum Dis 2015

RHEUMATOLOGY

Rheumatology 2016;55:2191–2199
doi:10.1093/rheumatology/kew326
Advance Access publication 19 September 2016

Original article

The influence of obesity on response to tumour necrosis factor-α inhibitors in psoriatic arthritis: results from the DANBIO and ICEBIO registries

Pil Højgaard[1,2], Bente Glintborg[1,3], Lars Erik Kristensen[2], Bjorn Gudbjornsson[4,5], Thorvardur Jon Love[5,6] and Lene Dreyer[1,2,7]

The median follow-up-time was 1.5 years

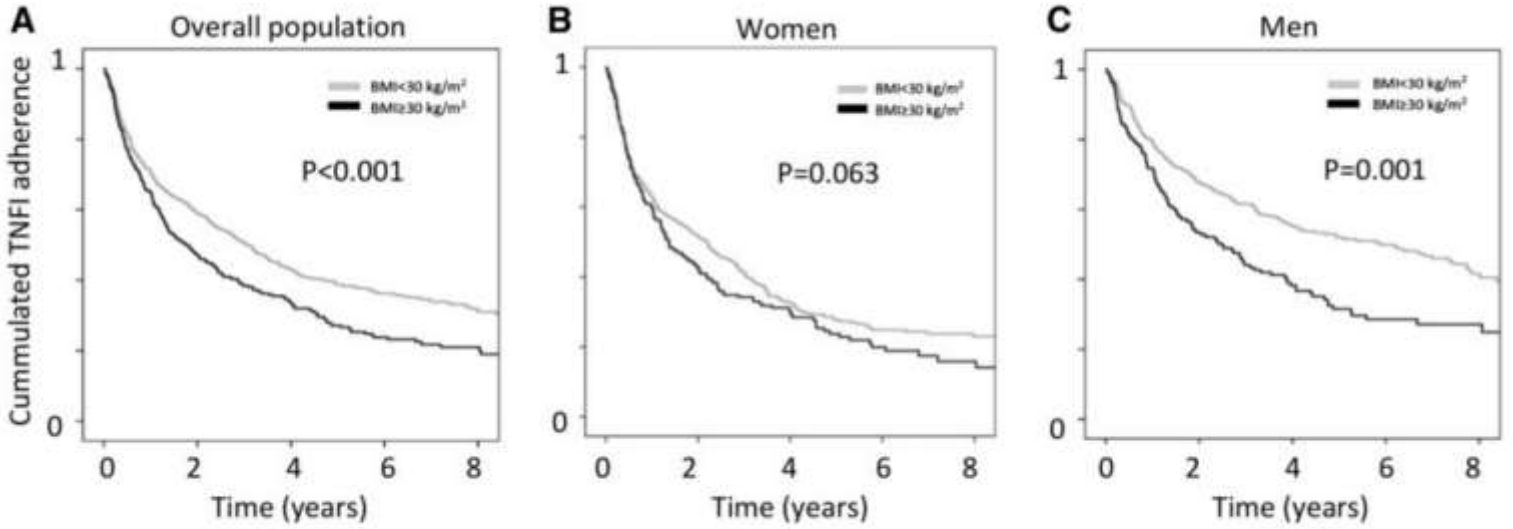
Obese patients had higher baseline disease activity:
28-joint DAS [mean 4.6 (S.D. 1.2) vs 4.4 (1.2)];
CRP [median 9 mg/l (IQR 519) vs 7 (318)];
visual analogue scale-pain [66 mm (IQR 48-76) vs 60 (38-74)]

TABLE 4 Impact of obesity on treatment response according to ACR and EULAR definitions

6 month response	Overall, OR (95% CI)	Women, OR (95% CI)	Men, OR (95% CI)	Adalimumab, OR (95% CI)	Infliximab, OR (95% CI)	Etanercept, OR (95% CI)
EULAR good, obese	0.75 (0.50, 1.15)	0.92 (0.50, 1.69)	0.53* (0.29, 0.99)	0.70 (0.37, 1.34)	0.59 (0.26, 1.37)	0.99 (0.31, 3.21)
EGOM[a], obese	0.47* (0.30, 0.74)	0.50* (0.27, 0.92)	0.36* (0.17, 0.79)	0.45* (0.21, 0.97)	0.39* (0.17, 0.92)	0.30 (0.09, 1.00)
ACR20, obese	0.65 (0.40, 1.06)	0.83 (0.41, 1.69)	0.40* (0.19, 0.86)	0.44* (0.21, 0.96)	0.76 (0.28, 2.02)	1.28 (0.39, 4.15)
ACR50, obese	0.71 (0.42, 1.18)	1.17 (0.53, 2.60)	0.43* (0.21, 0.91)	0.42* (0.19, 0.94)	0.97 (0.33, 2.82)	2.02 (0.51, 8.01)
ACR70, obese	0.80 (0.43, 1.48)	1.37 (0.46, 4.16)	0.59 (0.27, 1.30)	0.63 (0.24, 1.65)	1.35 (0.33, 5.52)	1.19 (0.15, 8.96)

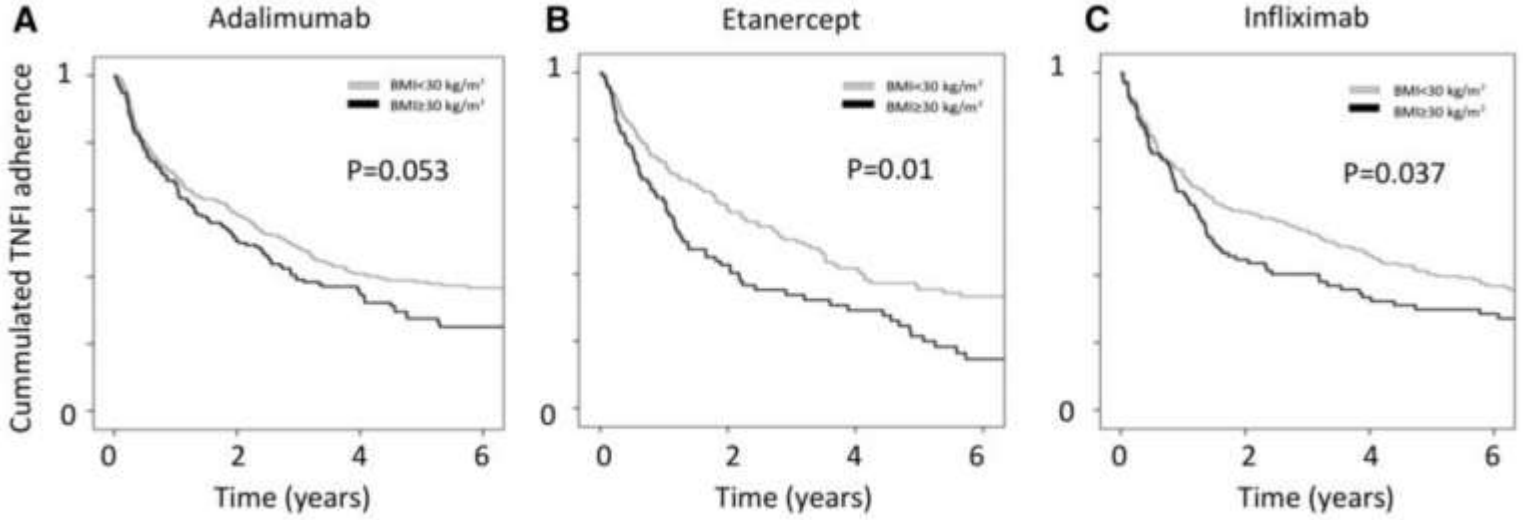
Results of multivariable logistic regression with adjustment for age (years), CRP (mg/l), DAS28, MTX (yes/no), smoking (yes/no), VAS-pain (0–100 mm), nationality (Danish/Icelandic), disease duration (years) and TNFI initiation year (2000–5/2006–15) as well as gender and TNFI drug types (all five). ORs and 95% CIs with non-obese (BMI <30) as reference (OR = 1). [a]EGOM: EULAR good or moderate response. *$P < 0.05$.

FIG. 1 Drug adherence in obese and non-obese patients overall and by gender



Kaplan–Meier plots of TNFI adherence in obese and non-obese patients depicted for (**A**) the overall population, (**B**) women and (**C**) men. *P*-values by log rank test.

FIG. 2 Drug adherence in obese and non-obese patients according to TNFI drug type



Kaplan–Meier plots of TNFI adherence in obese *vs* non-obese patients treated with (**A**) adalimumab, (**B**) etanercept and (**C**) infliximab. *P*-values by log rank test.

Obesity increased the risk of TNFI withdrawal [hazard ratio 1.6 (95% CI 1.3, 2.0)] and reduced odds for EGOM response [odds ratio 0.47 (95% CI 0.29, 0.72)].

Association of Body Mass Index Categories with Disease Activity and Radiographic Joint Damage in Rheumatoid Arthritis: A Systematic Review and Metaanalysis

Celine Vidal, Thomas Barnetche, Jacques Morel, Bernard Combe, and Claire Daïen

	Obese			Normal BMI				Mean Difference
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Fixed, 95% CI
Ajeganova 2013 [33]	5.4	1.1	206	5.2	1.2	775	59.3%	0.20 [0.03, 0.37]
Baker 2011 [31]	6.28	1.15	127	6.24	1.13	156	24.6%	0.04 [−0.23, 0.31]
Brown 2012 [34]	7.03	0.9	32	6.96	1	65	11.3%	0.07 [−0.33, 0.47]
Straburzyn'ska-Lupa 2013[32]	5.08	0.88	19	5.02	1.09	22	4.8%	0.06 [−0.54, 0.66]
Total (95% CI)			**384**			**1018**	**100.0%**	**0.14 [0.01, 0.27]**

Heterogeneity: $Chi^2 = 1.19$, $df = 3$ ($P = 0.76$); $I^2 = 0\%$
Test for overall effect: $Z = 2.06$ ($P = 0.04$)

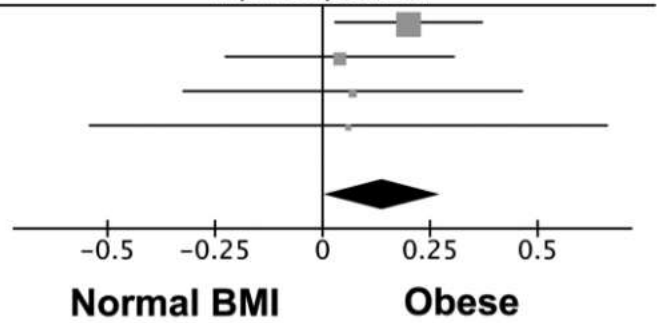


Figure 2. Forest plot of the effect of BMI (obesity ≥ 30 kg/m^2 vs normal weight 18.5–25 or 20–25 kg/m^2) on DAS28. BMI: body mass index; DAS28: Disease Activity Score in 28 joints; IV: independent variable.

Disease Activity Score in 28 joints (DAS28)

Vidal - J Rheumatol 2015

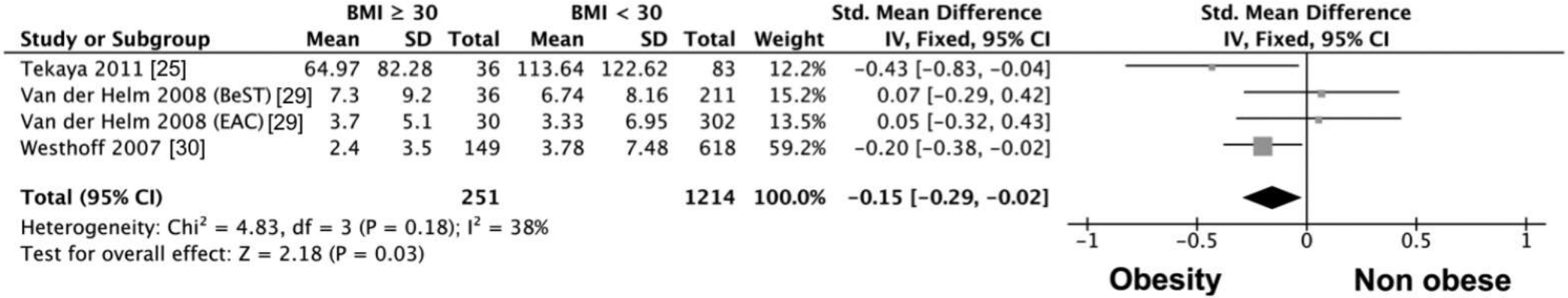


Figure 4. Forest plot of the effect of BMI (obesity ≥ 30 kg/m^2 vs non-obese < 30 kg/m^2) on radiographic joint damage (standardized means). BMI: body mass index; IV: independent variable.

Radiographic joint damage scored by Ratingen, Simple Erosion Narrowing Score (**SENS**), or modified Sharp/van der Heijde (**SvdH**) scales

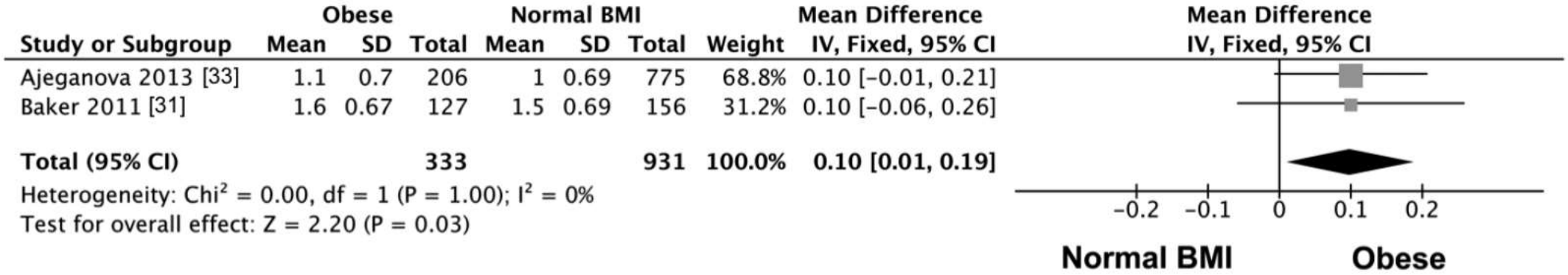


Figure 3. Forest plot of the effect of BMI (obesity ≥ 30 kg/m^2 vs normal weight 18.5–25 or 20–25 kg/m^2) on HAQ score. BMI: body mass index; HAQ: Health Assessment Questionnaire; IV: independent variable.

Health Assessment Questionnaire (**HAQ**)

Do we need new tools?

CMAJ | AUGUST 4, 2020 | VOLUME 192 | ISSUE 31

Serum adiponectin concentrations correlate with severity of rheumatoid arthritis evaluated by extent of joint destruction

Kosuke Ebina · Atsunori Fukuhara · Wataru Ando · Makoto Hirao · Tadashi Koga · Kazuya Oshima · Morihiro Matsuda · Kazuhisa Maeda · Tadashi Nakamura · Takahiro Ochi · Iichiro Shimomura · Hideki Yoshikawa · Jun Hashimoto

Table 4 Adjusted ORs of serum adiponectin level and BMI for disease severity of RA

	Adjusted OR	95% CI	*P* value
Adiponectin, μg/ml	1.085	1.007–1.168	0.031
BMI, kg/m^2	0.907	0.785–1.048	NS

ORs odds ratios, *95% CI* 95% confidence interval, *NS* not significant

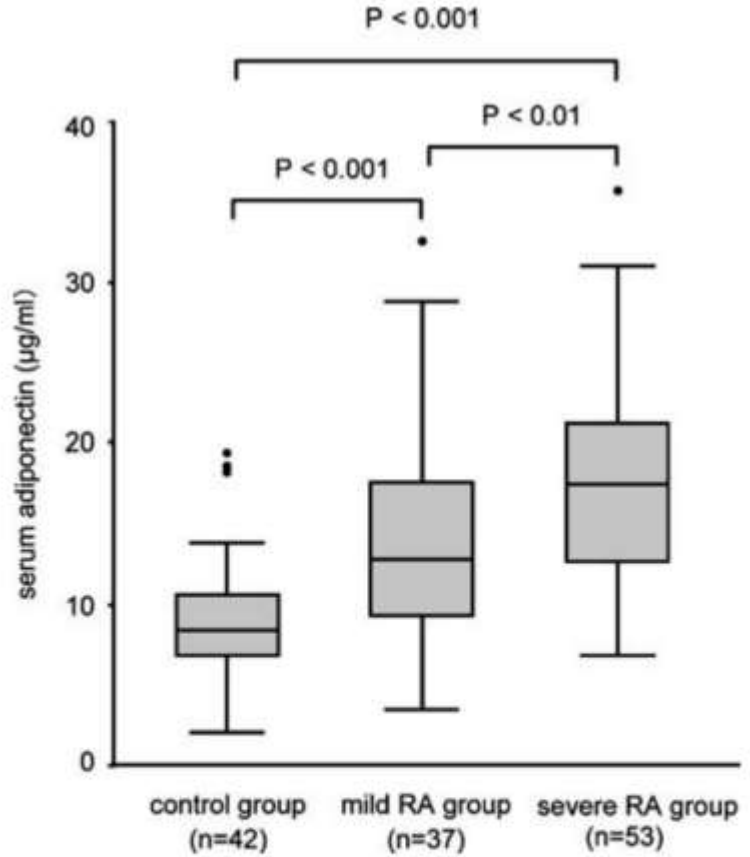
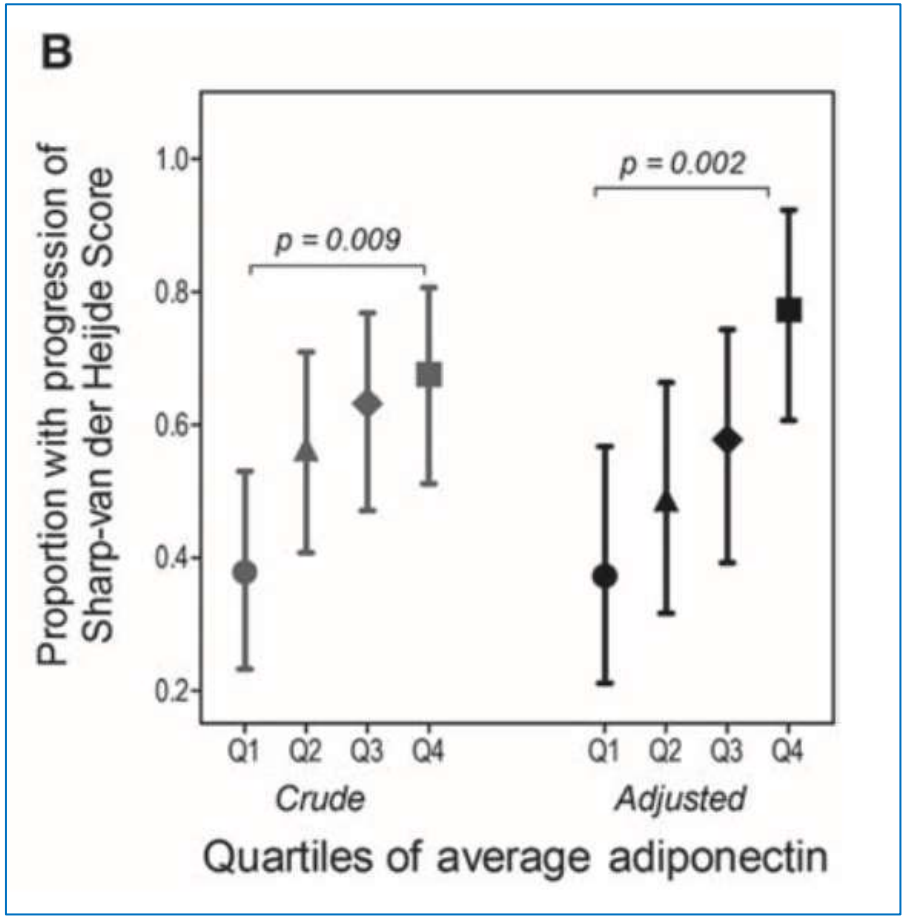


Fig. 1 Box-and-whisker plots of serum adiponectin levels in the control group, mild RA group, and severe RA group evaluated by the number of joint destruction in 68 joints on plain radiograph. The mean serum level of adiponectin was significantly higher in the severe RA group (17.7±6.7 μg/ml) than in the control (9.1±3.8 μg/ml) or mild RA group (13.9±6.5 μg/ml) (control vs. mild RA group: $P<0.001$, mild RA vs. severe RA group: $P<0.01$, control vs. severe RA group: $P<0.001$)

Ebina - Clin Rheumatol 2009

Association of circulating adiponectin levels with progression of radiographic joint destruction in rheumatoid arthritis

Jon T Giles,[1] Desiree M van der Heijde,[2] Joan M Bathon[1]



Giles - Ann Rheum Dis 2011

Increased circulating adiponectin is an independent disease activity marker in patients with rheumatoid arthritis: A cross-sectional study using the KURAMA database

Hiroto Minamino[1,2☯], Masao Katsushima[3☯], Tamami Yoshida[4], Motomu Hashimoto[5*], Yoshihito Fujita[1*], Mirei Shirakashi[3], Wataru Yamamoto[6], Kosaku Murakami[3], Koichi Murata[5], Kohei Nishitani[5], Masao Tanaka[5], Hiromu Ito[7], Nobuya Inagaki[1], Shuichi Matsuda[7]

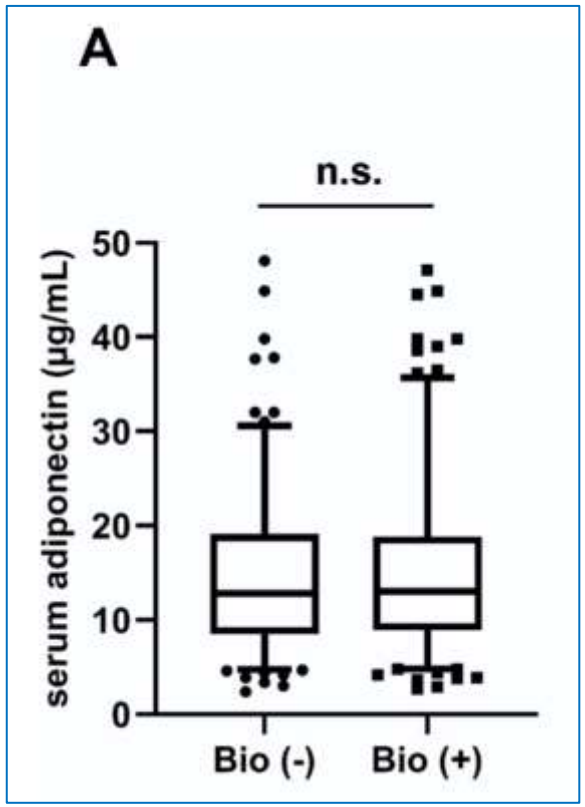


Table 3. Multiple regression analysis for independent factors associated with DAS28-ESR.

Dependent variables	Independent variables				95%CI	
		Estimates	Std. Error	*p*-value	Lower	Upper
DAS28-ESR	Prednisolone (+)	0.553	0.117	< .0001	0.323	0.782
	RF (1 IU/mL)	0.0007	0.00001	< .0001	0.00028	0.00099
	Age (10 years)	0.171	0.049	< .0001	0.075	0.27
	eGFR (10 ml/min/1.73m^2)	0.083	0.028	0.0033	0.028	0.14
	Sex (male)	-0.406	0.14	0.0037	-0.68	-0.13
	Adiponectin (1 μg/mL)	0.0127	0.0057	0.0258	0.0015	0.024
	Anti-CCP antibody (10 U/mL)	0.0025	0.0011	0.0259	0.0003	0.0047

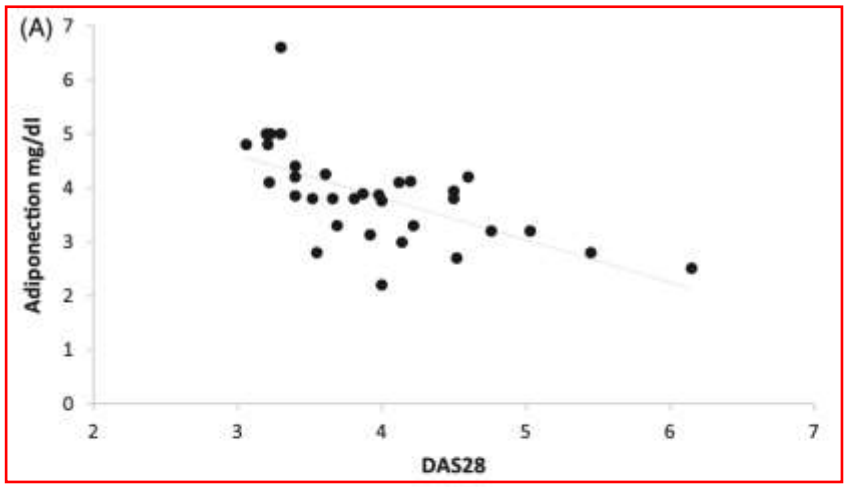
Covariates were selected from demographic, RA activity-related and life style-related factors: age, sex, body mass index, V/S ratio, eGFR, RA duration, RF, anti-CCP antibody, biological agent use, MTX use, PSL use, diabetes mellitus, hypertension, dyslipidemia, smoking habit and adiponectin. Units for estimates values are expressed in units in parentheses.

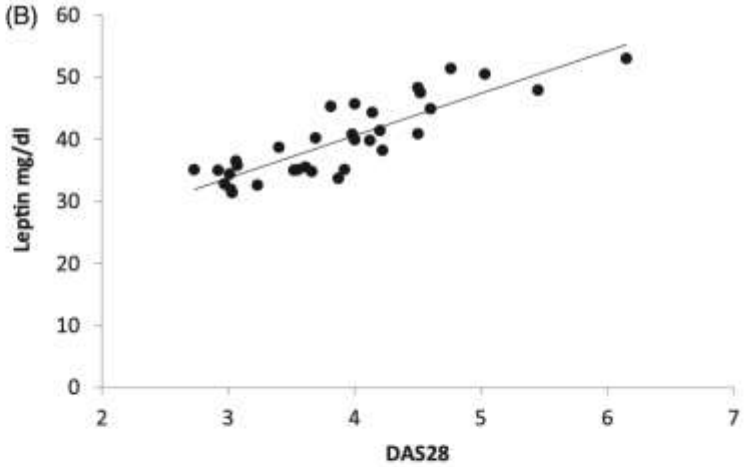
RF rheumatoid factor, *eGFR* estimated glomerular filtration rate, *anti-CCP antibody* anti-cyclic citrullinated peptide antibody.

Minamino - PlosOne 2020

Changes in serum adipokines profile and insulin resistance in patients with rheumatoid arthritis treated with anti-TNF-α

Addolorata Corrado, Ripalta Colia, Cinzia Rotondo, Eliana Sanpaolo and Francesco Paolo Cantatore





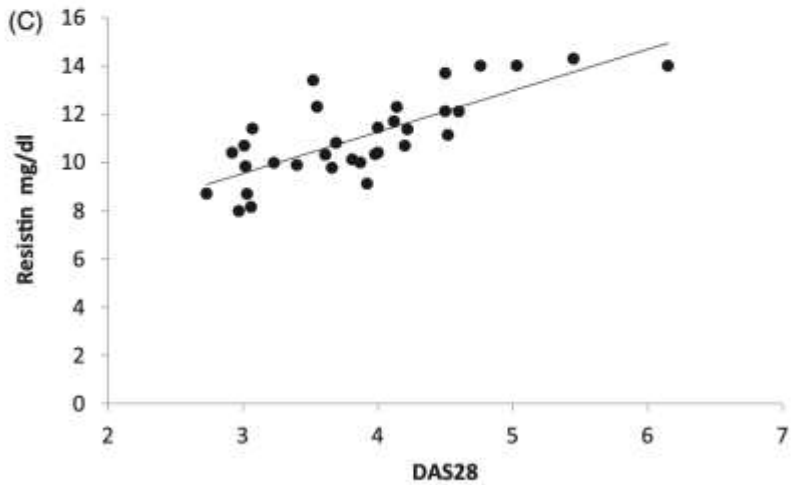


Figure 3. Correlation between disease activity and serum levels of leptin (a), resistin (b), and adiponectin (c) at baseline. An inverse relationship between disease activity (DAS28) and adiponectin levels is observed ($r = -0{,}68$; $p < 0.01$). Conversely serum levels of resistin ($r = 0.86$; $p < 0.001$) and leptin ($r = 0.86$; $p < 0.001$) directly correlate with DAS28.

Corrado - Curr Med Res Opin 2019

EULAR recommendations for cardiovascular disease risk management in patients with rheumatoid arthritis and other forms of inflammatory joint disorders: 2015/2016 update

eular
fighting rheumatic & musculoskeletal diseases together

Table 1 Overarching principles and recommendations

	Level of evidence	Strength of recommendation	Level of agreement (SD)
Overarching principles A. Clinicians should be aware of the higher risk for CVD in patients with RA compared with the general population. This may also apply to AS and PsA. B. The rheumatologist is responsible for CVD risk management in patients with RA and other IJD. C. The use of NSAIDs and corticosteroids should be in accordance with treatment-specific recommendations from EULAR and ASAS			
Recommendations			
1. Disease activity should be controlled optimally in order to lower CVD risk in all patients with RA, AS or PsA	2b-3	B	9.1 (1.3)
2. CVD risk assessment is recommended for all patients with RA, AS or PsA at least once every 5 years and should be reconsidered following major changes in antirheumatic therapy	3–4	C	8.8 (1.1)
3. CVD risk estimation for patients with RA, AS or PsA should be performed according to national guidelines and the SCORE CVD risk prediction model should be used if no national guideline is available	3–4	C–D	8.7 (2.1)
4. TC and HDLc should be used in CVD risk assessment in RA, AS and PsA and lipids should ideally be measured when disease activity is stable or in remission. Non-fasting lipids measurements are also perfectly acceptable	3	C	8.8 (1.2)
5. CVD risk prediction models should be adapted for patients with RA by a 1.5 multiplication factor, if this is not already included in the model	3–4	C	7.5 (2.2)
6. Screening for asymptomatic atherosclerotic plaques by use of carotid ultrasound may be considered as part of the CVD risk evaluation in patients with RA	3–4	C–D	5.7 (3.9)
7. Lifestyle recommendations should emphasise the benefits of a healthy diet, regular exercise and smoking cessation for all patients	3	C	9.8 (0.3)
8. CVD risk management should be carried out according to national guidelines in RA, AS or PsA, antihypertensives and statins may be used as in the general population	3–4	C–D	9.2 (1.3)
9. Prescription of NSAIDs in RA and PsA should be with caution, especially for patients with documented CVD or in the presence of CVD risk factors	2a-3	C	8.9 (2.1)
10. Corticosteroids: for prolonged treatment, the glucocorticoid dosage should be kept to a minimum and a glucocorticoid taper should be attempted in case of remission or low disease activity; the reasons to continue glucocorticoid therapy should be regularly checked	3–4	C	9.5 (0.7)

AS, ankylosing spondylitis; ASAS, Assessment of Spondyloarthritis International Society; CVD, cardiovascular disease; EULAR, European League against Rheumatism; HDLc, high-density lipoprotein cholesterol; IJD, inflammatory joint disorder; NSAID, non-steroidal anti-inflammatory drug; PsA, psoriatic arthritis; RA, rheumatoid arthritis; SCORE, Systematic Coronary Risk Evaluation; TC, total cholesterol.

Overarching prinicples

A. Clinicians should be aware of the higher risk for CVD in patients with RA compared with the general population. This may also apply to AS and PsA.

B. The rheumatologist is responsible for CVD risk management in patients with RA and other IJD.

Important to note the responsibility concerns that's gets done, but not that this should be done by rheumatologists themselves!

1. **CONSAPEVOLEZZA**
2. **GESTIONE MULTIDISCIPLINARE**

ESC European Society of Cardiology

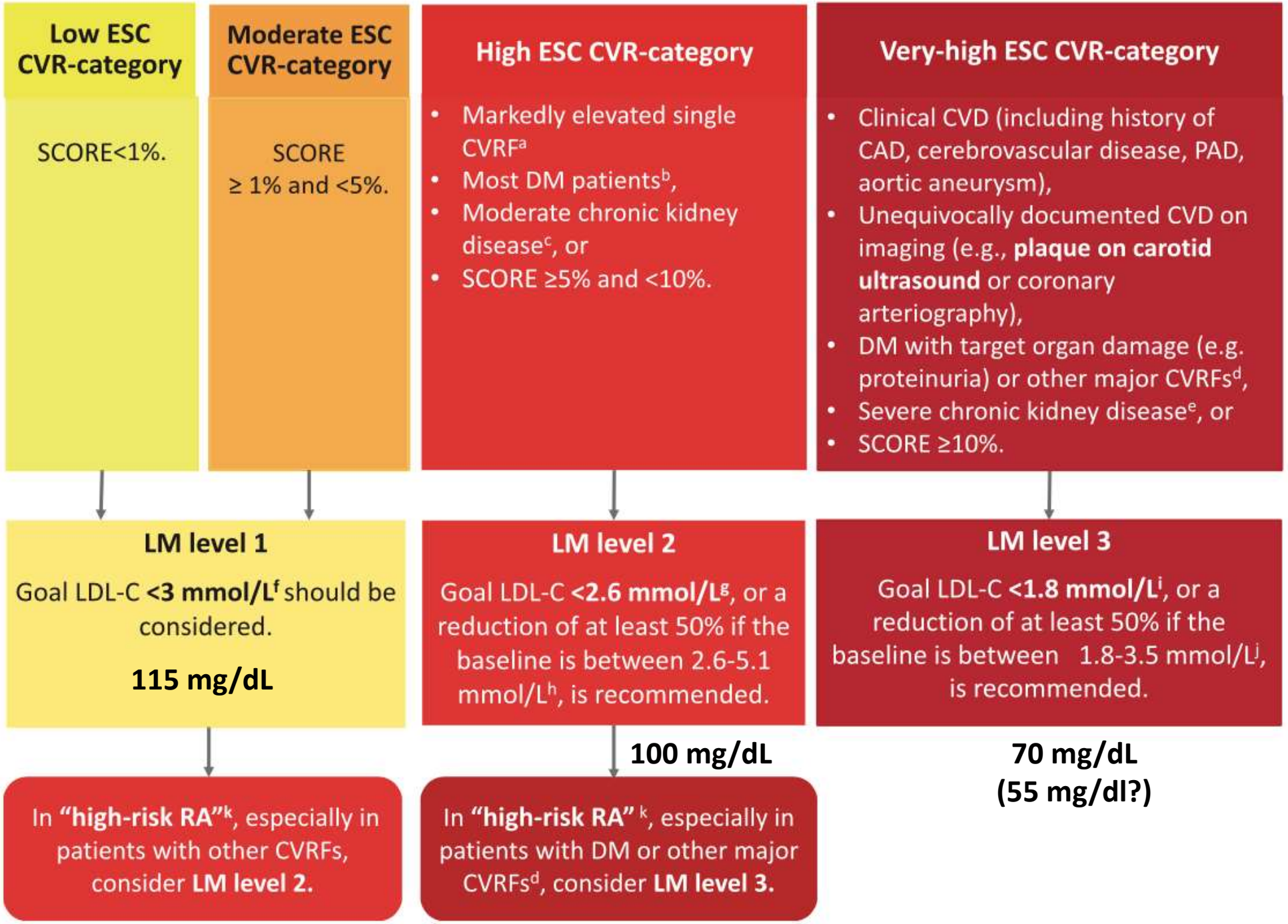
European Heart Journal - Cardiovascular Pharmacotherapy (2020) **6**, 104–114
doi:10.1093/ehjcvp/pvz033

Lipid management in rheumatoid arthritis: a position paper of the Working Group on Cardiovascular Pharmacotherapy of the European Society of Cardiology

Ivana Hollan [1,2]*, Nicoletta Ronda[3], Patrick Dessein[4,5,6], Stefan Agewall[7], George Karpouzas[8], Juan Tamargo[9], Alexander Niessner[10], Gianluigi Savarese[11], Giuseppe Rosano[12], Juan Carlos Kaski[13], Sven Wassmann[14,15], and Pier Luigi Meroni[16]

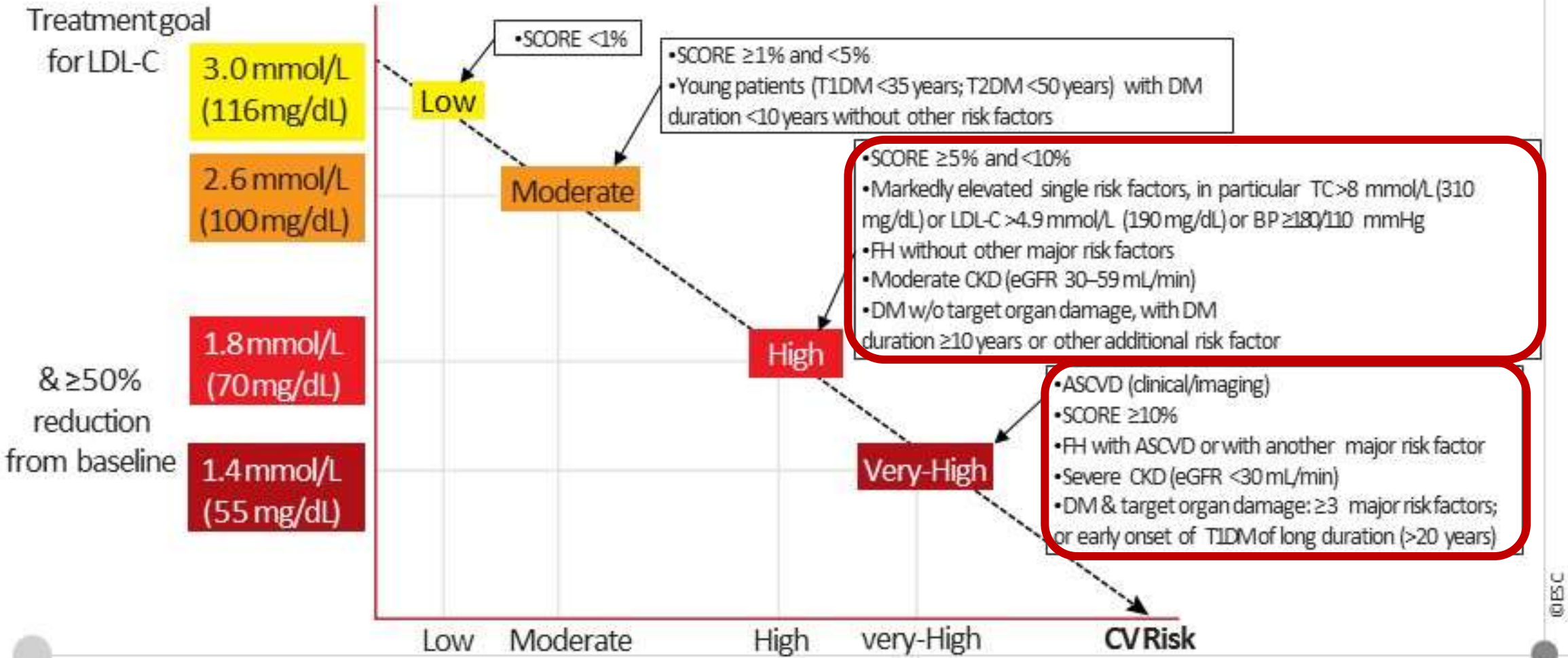
"Low-risk RA" is defined as:

- Seronegative
- non-erosive
- in patients without extra-articular manifestations
- in long-term (>1 year) remission (CDAI <_2.8 or SDAI <_3.3 or DAS28-ESR<_2.6)
- without acute arthritis or persistently elevated acute phase reactants (C-reactive protein or erythrocyte sedimentation rate)
- with well-preserved physical function (HAQ-DI <_0.5)
- without high cumulative disease activity
- not currently using glucocorticoids and without high cumulative glucocorticoid dose



Central Illustration Upper panel Treatment goals for low-density lipoprotein cholesterol (LDL-C) across categories of total cardiovascular disease risk

EAS ESC European Society of Cardiology



www.escardio.org/guidelines

2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk (European Heart Journal 2019 -doi: 10.1093/eurheartj/ehz455)

- We propose a new algorithm for CVR estimation/LM (*Figure 1*).
  - Patients with 'low-risk RA' (seronegative, non-erosive RA, without extra-articular manifestations, in long-term remission, with well-preserved physical function, not currently using glucocorticoids, and without high cumulative disease activity and high cumulative glucocorticoid dose) should follow LM recommendations for the general population, but LDL-C<3 mmol/L (115 mg/dL) should be considered in all individuals from the low/moderate CVR-categories. 'High-risk RA' reclassifies patients into a higher ESC CVR-category, requiring lower LDL-C targets than those recommended for the general population.
    - Thus, RA patients should ideally have LDL-C levels <3 mmol/L (115 mg/dL), but many (including 'high-risk RA') <2.6 mmol/L (100 mg/dL), and some <1.8 mmol/L (70 mg/dL).

- All diabetics >40 years of age should use statins. This treatment may be considered also in younger diabetics with pronounced CVR.[30]
- In order to correctly treat patients with very-high CVR, and given the tendency to atypical clinical CVD picture in RA, proactive approach to diagnosing CVD is critical.
- Ultrasonographic detection of carotid plaques can facilitate determining very-high CVR and may be particularly important in 'high-risk RA'. It can be meaningful in low to high ESC CVR-categories.
- The overall situation, including comorbidities, treatment, lifestyle, and socioeconomic status, should be considered in CVR estimation.

Lipid assessment

- Lipid monitoring in RA should include TC, LDL-C, HDL-C, and TG levels and can be performed under non-fasting conditions. If non-fasting TGs ≥2.3 mmol/L (200 mg/dL), fasting TG assessment should be performed.
- Lp(a) screening should be considered.
- Non-HDL-C may be superior to LDL-C as CVR marker, especially in patients with high TG and low LDL-C, and in non-fasting samples.
- We recommend lipid assessment in RA regardless of age, at least every 5 years in 'low-risk RA', and annually in 'high-risk RA'.
- More frequent assessment should be considered in patients with severe lipid abnormalities and poor therapeutic response, rapidly progressing RA or CVR estimate close to thresholds mandating lower LDL-C targets.
- Reassessment is indicated after changes significantly influencing CVR (e.g. lifestyle modifications or initiation of DMARD or high-dose glucocorticoid treatment).

Therapeutic interventions

- RA patients should receive adequate counselling and support regarding diet, physical activity, and other beneficial lifestyle modifications.
- Pharmacological treatment of hypercholesterolaemia should be performed primarily by statins.
  - The optimal form of statin regimen in RA is unknown, but statins with profound anti-inflammatory effects (e.g. atorvastatin or rosuvastatin) may be particularly beneficial.
- If goal LDL-C cannot be reached through lifestyle and statins, other LMTs (PCSK9, ezetimibe, and fibrates) should be considered, following general recommendations.
- Treatment of other lipid aberrations, including high TG and Lp(a) levels, should follow general recommendations.
- Control of RA activity may ameliorate some alterations of lipid homeostasis, (e.g., decrease Lp(a) levels and improve lipoprotein functions and cellular cholesterol transport), and decrease overall CVR.

Implementation of LM

- LM in RA can be administered by general practitioners, in collaboration with, e.g. cardiologists, endocrinologists, lipidologists, dietitians, nurses, and physical therapists Rheumatologists should take the lead and ensure that adequate LM is provided.
- There should be focus on education of patients and health care providers about CVR/LM in RA.

Intensity of lipid lowering treatment

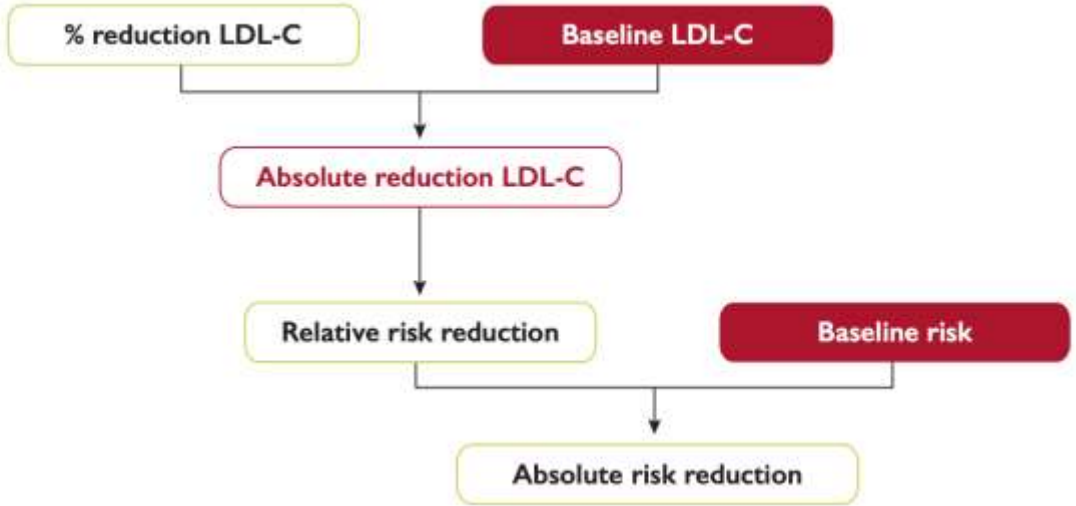
Treatment	Average LDL-C reduction
Moderate intensity statin	≈ 30%
High intensity statin	≈ 50%
High intensity statin plus ezetimibe	≈ 65%
PCSK9 inhibitor	≈ 60%
PCSK9 inhibitor plus high intensity statin	≈ 75%
PCSK9 inhibitor plus high intensity statin plus ezetimibe	≈ 85%

ESC European Society of Cardiology
European Heart Journal (2019) **00**, 1–78
doi:10.1093/eurheartj/ehz455

ESC/EAS GUIDELINES

2019 ESC/EAS Guidelines for the management of dyslipidaemias: *lipid modification to reduce cardiovascular risk*

The Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS)

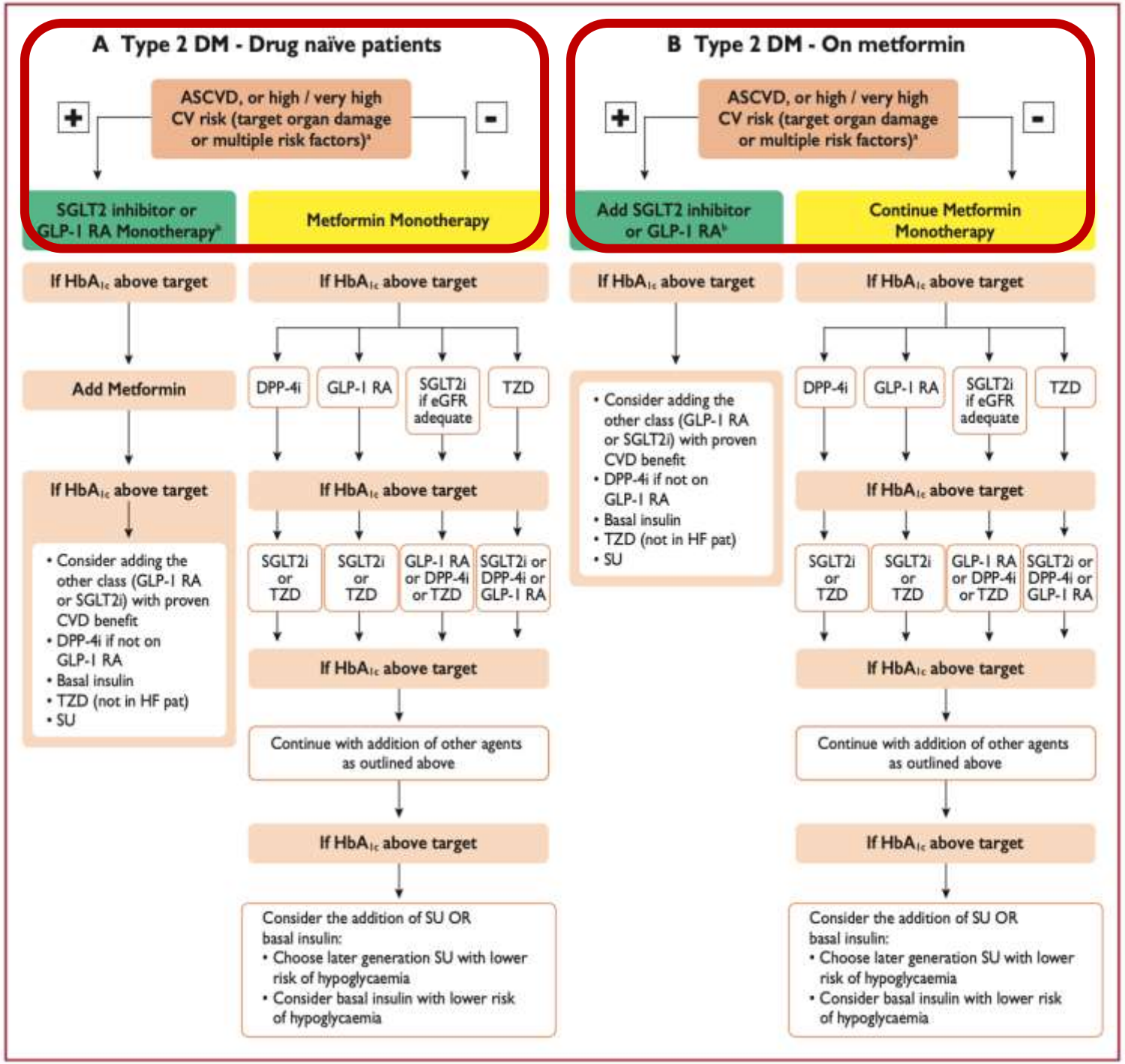


ESC European Society of Cardiology
European Heart Journal (2019) 00, 1–69
doi:10.1093/eurheartj/ehz486

ESC GUIDELINES

2019 ESC Guidelines on diabetes, pre-diabetes, and cardiovascular diseases developed in collaboration with the EASD

The Task Force for diabetes, pre-diabetes, and cardiovascular diseases of the European Society of Cardiology (ESC) and the European Association for the Study of Diabetes (EASD)



Cardiovascular Actions and Clinical Outcomes With Glucagon-Like Peptide-1 Receptor Agonists and Dipeptidyl Peptidase-4 Inhibitors

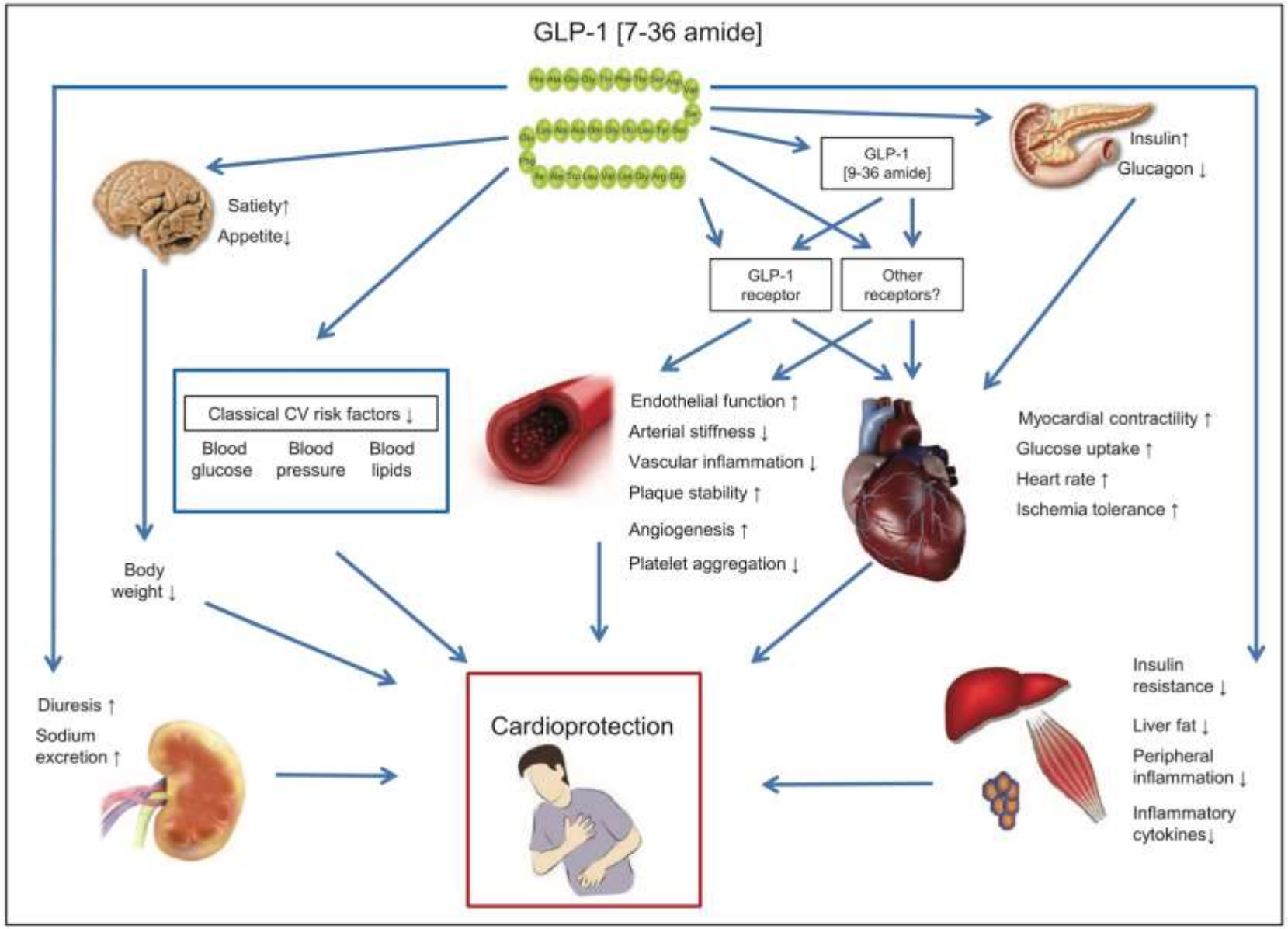


Figure 3. Potential mechanisms mediating a beneficial effect of glucagon-like peptide-1 (GLP-1) receptor agonists on reducing cardiovascular events.

Effects of diabetes mellitus-related parameters (glycemic control, avoidance of [severe] hypoglycemia), cardiovascular risk factors (body weight, blood pressure, lipoproteins/lipids), and interactions with GLP-1 receptors in the cardiovascular system (potentially leading to improved endothelial function/vasodilation, improved cardiac function under conditions of coronary ischemia, and anti-inflammatory/ anti-atherosclerotic effects) have to be considered. CV indicates cardiovascular.

Nauck - Circulation 2017

CONCLUSIONI

- CONSAPEVOLEZZA del **rischio CV (↑ nelle artropatie infiammatorie)**
- **Fattori metabolici (adiposità) nelle artropatie infiammatorie**: valore predittivo di risposta e aderenza alla terapia + valore prognostico
- **Quale marker di adiposità?** BMI vs. adipochine
- **Nuovo approccio alla stima del rischio CV** per artrite reumatoide
- **Controllo dei fattori cardio-endocrino-metabolici**: obiettivi personalizzati (es. LDL-C < 115 mg/dl o <100 / 70 mg/dl) e disponibilità di terapie innovative e sicure
- **Gestione MULTIDISCIPLINARE** (ruolo del *team*)

TORINO
16-17 ottobre 2020

Grazie per l’attenzione